input, modulating intrinsic mechanical properties in response to signal transduction and reciprocally modulating signal transduction in accordance with these mechanical properties. However, *in vivo* evidence that the cytoskeleton carries out similar functions during embryonic cell fate specification remains limited. Here we show a critical, *in vivo* role for the cytoskeleton in modulating heart precursor cell specification. In the basal chordate *Ciona intestinalis*, heart founder cells divide asymmetrically. The smaller daughters undergo differential induction to form the heart precursor lineage. Through staged dissociations, we show that cell–cell contact mediates differential induction at a specific developmental timepoint. At this time, heart founder cells form a polarized, highly invasive membrane which penetrates the underlying epidermis. Through targeted manipulations of Cdc42 activity and actin dynamics we demonstrate that these polarized protrusions serve to spatially restrict inductive signaling. These findings illustrate the importance of bi-directional interactions between intercellular signaling and the cytoskeleton during embryonic development. These studies also highlight the potential for dynamic cytoskeletal changes to refine cell fate specification in response to crude morphogen gradients.

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Program/Abstract # 36
Mechanisms of trophectoderm fate specification in preimplantation mouse embryos
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Preimplantation mouse embryos form two types of cells by the blastocyst stage: trophectoderm and inner cell mass. Historically, two models have been proposed for the mechanisms of cell fate specification. One is the Inside–Outside model, in which cell position determines cell fate, and the other is the Polarity model, in which the presence or absence of apico-basal polarity in cells controls cell fate. We previously showed that differential Hippo signaling along the inside–outside axis regulates cell fate by modulating activity of the transcription factor, Tead4. Our current goal is to integrate the molecular mechanism of cell fate regulation by the Hippo pathway with the two historical models. We found that manipulating cell positioning altered Hippo signaling and cell fates. Interestingly, manipulating cell polarity also affected Hippo signaling and cell fates. These results suggest that both Inside–Outside and Polarity models operate in preimplantation embryos, and that both mechanisms control cell fates via Hippo signaling pathway. I will discuss how cell position-dependent and cell polarity-dependent mechanisms operate in preimplantation embryos and how they modulate Hippo signaling to control cell fates.

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Program/Abstract # 37
Expression of Oct4, Cdx2 and Yap1 during blastocyst formation in the marsupial, *Monodelphis domestica*
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The marsupial blastocyst first forms as a simple epithelium securely adherent to the zona pellucida. It has no inner cell mass (ICM), which in the eutherian (mouse) blastocyst contains the embryonic stem cells at this stage. The marsupial ICM equivalent, the pluriblast, is co-planar with the trophoblast. By contrast, the mouse trophoblast encloses the ICM. Oct4, Cdx2 and Yap1 play crucial roles in allocating mouse blastocyst cells to either trophoblast or ICM fates. Because these genes are found in the opossum genome, we hypothesized that they may have a similar role in cell allocation between pluriblast and trophoblast in the opossum blastocyst. In both mouse and opossum, Oct4 is expressed in all embryonic cells prior to blastocyst formation. During mouse blastocyst formation, Cdx2 is upregulated as Oct4 is downregulated in the nascent trophoblast. In the opossum, a patch of cells in the unilaminar blastocyst epithelium (pluriblast) undergoes the same switch in gene expression. In both types of embryos, Yap1 is translocated to the nuclei of putative trophoblast cells but remains in the cytoplasm of cells fated to be ICM or pluriblast. Our results indicate that the roles of Oct4, Cdx2 and Yap1 in allocating cells to the trophoblast lineage are evolutionarily conserved between marsupials and eutherians, despite the topological differences between their blastocysts. Our results also suggest that cell position plays less of a role in mammalian trophoblast differentiation as has been long believed.

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Program/Abstract # 38
Communication between nonadjacent blastomeres in early *Xenopus* embryos
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Confocal microscopy of surgically opened *Xenopus* embryos expressing membrane-tethered eGFP unexpectedly revealed hundreds of extremely long, stable membranous processes traversing the entire volume of the blastocoel, linking blastomeres as far as 300 µm apart. Most blastomeres appear to be in long-term contact with a dozen or more adjacent and nonadjacent cells via these long filopodia. The processes are filled with f-actin, and small cytoplasm-bearing blebs translocate bidirectionally within them. They are not cytoplasmic bridges and are not produced during cell division; rather they develop via filopodial extension, exploration and contact. The longest filopodia observed occur before the 512-cell stage, a period critical for embryonic patterning in *Xenopus*. By early blastula stage, protrusive activity abruptly subsides: cells facing the blastocoel display only short protrusions and contact only their immediate neighbors. During this transition, the filopodia settle onto nearby cell surfaces and break up into chains of membrane-bound vesicles. The timing and distribution of these remarkable structures suggest vectorial transport of maternal components between nonadjacent cells. In support of this idea, we observed transfer of lucifer yellow-labeled cytoplasm between nonadjacent blastomeres via engulfment of vesicles budded from the long processes during their breakup. We are presently exploring whether 1) the spatial pattern of traversing filopodia is biased with respect to the primary embryonic axes; 2) early perturbation of dorsal–ventral patterning alters their deployment; or conversely 3) their disruption affects embryonic patterning.
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